

GRB10 Blocking Peptide (N-term)

Synthetic peptide Catalog # BP20489a

Specification

GRB10 Blocking Peptide (N-term) - Product Information

Primary Accession

013322

GRB10 Blocking Peptide (N-term) - Additional Information

Gene ID 2887

Other Names

Growth factor receptor-bound protein 10, GRB10 adapter protein, Insulin receptor-binding protein Grb-IR, GRB10, GRBIR, KIAA0207

Target/Specificity

The synthetic peptide sequence is selected from aa 16-30 of Human GRB10

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

GRB10 Blocking Peptide (N-term) - Protein Information

Name GRB10

Synonyms GRBIR, KIAA0207

Function

Adapter protein which modulates coupling of a number of cell surface receptor kinases with specific signaling pathways. Binds to, and suppress signals from, activated receptors tyrosine kinases, including the insulin (INSR) and insulin-like growth factor (IGF1R) receptors. The inhibitory effect can be achieved by 2 mechanisms: interference with the signaling pathway and increased receptor degradation. Delays and reduces AKT1 phosphorylation in response to insulin stimulation. Blocks association between INSR and IRS1 and IRS2 and prevents insulin-stimulated IRS1 and IRS2 tyrosine phosphorylation. Recruits NEDD4 to IGF1R, leading to IGF1R ubiquitination, increased internalization and degradation by both the proteasomal and lysosomal pathways. May play a role in mediating insulin-stimulated ubiquitination of INSR, leading to proteasomal degradation. Negatively regulates Wnt signaling by interacting with LRP6 intracellular portion and interfering with the binding of AXIN1 to LRP6. Positive regulator of the KDR/VEGFR-2 signaling pathway. May inhibit NEDD4-mediated degradation of KDR/VEGFR-2.



Cellular Location

Cytoplasm. Note=When complexed with NEDD4 and IGF1R, follows IGF1R internalization, remaining associated with early endosomes. Uncouples from IGF1R-containing endosomes before the sorting of the receptor to the lysosomal compartment (By similarity).

Tissue Location

Widely expressed in fetal and adult tissues, including fetal and postnatal liver, lung, kidney, skeletal muscle, heart, spleen, skin and brain.

GRB10 Blocking Peptide (N-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

• Blocking Peptides

GRB10 Blocking Peptide (N-term) - Images

GRB10 Blocking Peptide (N-term) - Background

Adapter protein which modulates coupling of a number of cell surface receptor kinases with specific signaling pathways. Binds to, and suppress signals from, activated receptors tyrosine kinases, including the insulin (INSR) and insulin-like growth factor (IGF1R) receptors. The inhibitory effect can be achieved by 2 mechanisms: interference with the signaling pathway and increased receptor degradation. Delays and reduces AKT1 phosphorylation in response to insulin stimulation. Blocks association between INSR and IRS1 and IRS2 and prevents insulin-stimulated IRS1 and IRS2 tyrosine phosphorylation. Recruits NEDD4 to IGF1R, leading to IGF1R ubiquitination, increased internalization and degradation by both the proteasomal and lysosomal pathways. May play a role in mediating insulin-stimulated ubiquitination of INSR, leading to proteasomal degradation. Negatively regulates Wnt signaling by interacting with LRP6 intracellular portion and interfering with the binding of AXIN1 to LRP6. Positive regulator of the KDR/VEGFR-2 signaling pathway. May inhibit NEDD4-mediated degradation of KDR/VEGFR-2.

GRB10 Blocking Peptide (N-term) - References

Liu F., et al. Proc. Natl. Acad. Sci. U.S.A. 92:10287-10291(1995). O'Neill T.J., et al. J. Biol. Chem. 271:22506-22513(1996). Frantz J.D., et al. J. Biol. Chem. 272:2659-2667(1997). Dong L.Q., et al. J. Biol. Chem. 272:29104-29112(1997). Nantel A., et al. J. Biol. Chem. 273:10475-10484(1998).